ON THE RELATIONS OF INFANTILE SPINAL PARALYSIS TO SPINAL DISEASES OF LATER LIFE.*

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The fact that infantile spinal paralysis may give rise to spinal diseases in later life, as long as from thirty to fifty years after the occurrence of the acute poliomyelitis anterior was first recognized by a number of French authors. Ballet, Dutil, E. Thomas, Sattler, and Charcot have reported cases of this kind, and recently similar cases have been published by M. Laehr, A. Dutil, Fibroy, and Langer. Altogether, nine cases are recorded. An autopsy has not been made in any case. As to the clinical symptoms, they indicate in all cases an exclusive affection of the motor neurons, sensory symptoms having been entirely absent, so that in every case the diagnosis of progressive muscular atrophy, mostly of the type of Aran-Duchenne, could be made. The only exception to this is in the case of Laehr, in which the patient complained of pains in the knee-joint and shoulder-joint, and also had difficulty in urinating. The latter symptom, however, Laehr refers to a hypertrophy of the prostatic gland; and the pain in the joints is not a very rare symptom of progressive muscular atrophy, so that according to its clinical aspect, this case also could be placed in the same category.

There is, however, an interesting difference among these cases as to their relation to the previous infantile paralysis. In some cases (Dutil, Thomas, Charcot, and Laehr), the muscular atrophy began in parts which had not been affected by the previous disease, but were even anatomically quite remote from the previously affected parts. In Charcot's case, for instance, the infantile paralysis affected the lower extremities, mostly the left leg, while the later disease began in the muscles of the right shoulder. In other cases the atrophy of the later disease started in the previously affected parts as well as in the healthy ones. In one case of Dutil, for instance, the poliomyelitis had affected the right arm and left leg, while thirty-six years later the muscular atrophy appeared in both upper and lower extremities. The third class of cases seems to be represented by Langer's case, in which the poliomyelitis had affected both right extremities and a slight portion of the left leg, and in which the later disease was strictly confined to the muscles which had been attacked previously by the infantile paralysis.

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Various theories have been offered as to the nosological relation between the early infantile paralysis and the later disease. According to some authors, irritation of the cord by the old lesion enfeebled this organ and made it a *locus minoris resistentiæ*, which on any occasion might become susceptible to further disease. Charcot expressed the view that there existed in some individuals a certain disposition, a kind of vulnerability of the ganglionic cells of the anterior horns, which at some period of life might give rise to an acute poliomyelitis anterior, and at another to a progressive muscular atrophy, so that in fact both diseases would form different periods of one and the same pathological process. The third view was that the old scar, which was produced by the inflammatory process in the gray matter of the anterior horns, formed a latent but permanent inflammatory focus, which at any time might light up afresh and cause a new set of symptoms. Besides Charcot, Strümpell and others, Ballet and Dutil offered this theory, which they expressed in the title of their pamphlet: "Des quelques accidents spinaux determinés par la présence dans la moelle d'un ancien foyer de myélite infantile."

It is evident that in view of the difference of the clinical aspect of these cases which I described above, any of these theories might hold good for some particular case. This, however, could only be ascertained by a post-mortem examination, which, so far as I know, has not yet been made in any of these cases. The following is a case in which I was fortunate enough to get an autopsy:

M. D., a tailor, forty-five years old, presented himself at my clinic in October, 1895. He denied having had syphilis. There were no nervous or mental diseases in his family. He was married twice; neither of his wives had ever had an abortion. He had two healthy children. He was in perfect health up to three years ago, when he first noticed a stiffness and weakness in his left leg, which gradually became worse. After some time the right leg also became affected. He soon became unable to use his legs in working his sewing machine, and his gait became so much impaired that he was not able to stand or walk for any length of time. Besides this affection of the lower extremities the patient had nothing to complain of at that time. He stated that the use of the upper extremities was not impaired. There were no sensory symptoms whatsoever, nor was there a disturbance in the functions of the bladder and rectum. His sexual power was not diminished.

The objective examination revealed an atrophic condition of some of the muscles of the trunk and the upper extremities. The deltoid, the supraspinatus and infraspinatus of the right side showed quite a marked atrophy. There was fibrillation in the muscles of the trunk and shoulder. There was also diminished electric excitability, and in some muscles (deltoid and supraspinatus) reaction of degeneration. There was no atrophy in the muscles of the lower extremities, and the electric reaction was perfectly normal in these parts. The tendon reflexes of the lower extremities were considerably increased. The knee-jerks were extremely exaggerated, and there was bilateral ankle-clonus. The latter very frequently came on spontaneously, to the great annoyance of the patient. The tendon-of-Achilles reflex was increased. The superficial reflexes, like the cremaster, the abdominal and plantar, were present. The reflexes of the upper extremities, of the triceps, were not increased. Sensation to touch was perfectly normal in all parts of the body, as well as the sense of pain and temperature. The muscular sense was not impaired. There were no abnormal conditions in the functions of the cranial nerves, except that the tongue was slightly atrophied, and there was a marked masseter-reflex. Speech was in no way affected. There was no nystagmus. The reaction of the pupils, both to light and accommodation, was normal. The ophthalmoscopical examination revealed nothing abnormal.

Apart from this condition, there existed a very marked atrophy of the muscles of the left shoulder and upper arm. The deltoid had almost entirely disappeared. The biceps and triceps were reduced to a minimum, and the supraspinatus and infraspinatus, as well as the pectoral muscles, were also atrophic in the highest degree. He could, however, perform all motions with this extremity, though in a less degree.

When the patient came to me he did not mention this affection, because it had existed, as he said, as long as he could remember, since his earliest childhood. From the present state and from the history of this affection, as it could be learned through his relatives, it became evident that as a child of about two years he had an attack of infantile spinal paralysis, leaving behind the affection of the left upper extremity.

In this condition I presented the patient to the New York Neurological Society at the meeting of October 1, 1895. I maintained at that time that it could be proved by the history of the clinical symptoms that the pathological process of the later disease started from the place where the old scar of the poliomyelitis anterior was located. This lesion lay apparently in the left anterior horn of the cervical region of the cord. I stated at that time that as the man had first noticed weakness and stiffness of the left leg, the process must have approached first the left pyramidal tract which lay next to the scar, then after the affection of the right horn, which showed itself in atrophy of the muscles of the right upper extremity, the pathological process must have extended to the right pyramidal tract, causing a spastic condition in the right leg. According to this, we had an affection of both anterior horns and both pyramidal tracts in the cervical region of the cord, and, therefore, I called the case one of amyotrophic lateral sclerosis.

The further development of the clinical symptoms was as follows: The spastic condition of the lower extremities became gradually more and more intense, so that after three months, in January, 1896, the man was not able to walk at all. At this time he began to complain of very severe shooting pains in both lower extremities, which gradually became so intense that he had to be given hypodermic injections of morphine at frequent intervals. From the month of March he was confined to his bed. He was no longer able to sit up in bed or even to raise his head. Both lower extremities were in a permanent position of flexion and adduction. The spastic adduction was so intense that he had to wear a cushion beween the two knees in order to overcome the severe pressure. The rigidity of the limbs was such that they could not even forcibly be abducted or extended. The shooting pains grew worse and worse, while the atrophy of the muscles of the lower extremities made no further progress during the last six months. The paralysis of both upper extremities, however, became gradually worse until the patient could not move the left extremity at all, with the exception of some slight flexion of the fingers. On the right side the arm was also completely paralyzed, but the movements of the hand seemed unimpaired during the whole time of his disease. Sensation was perfectly normal. The lower extremities were even somewhat hypersensitive. The function of the bladder and rectum had remained normal during the whole time.

In April he began to experience some difficulty in swallowing, which gradually became more marked until towards the end of May he had become unable to swallow at all, so that he had to be fed with the tube. Without developing any other symptoms he died on the 14th of June, 1897.

A post-mortem examination was made about eight hours after death. On account of the great difficulties under which the autopsy had to be made, the internal organs and the peripheral nerves could not be examined. Only the brain and the spinal cord were obtained. The skull was symmetrical and showed no abnormalities. There were no adhesions between it and the dura. The sinuses were filled with blood. The pia appeared hyperemic, but could be removed from the cortex without difficulty. The convexity of the hemispheres was symmetrical. The central convolutions showed no difference in size on both sides. Cross-sections through the medulla oblongata revealed macroscopically nothing abnormal. The spinal cord, however, showed indistinct outlines of the gray substance at various places. The consistency was normal, while the color seemed to be a little more reddish than under normal circumstances. Brain, medulla and cord were hardened in a 5 per cent. formalin solution, and small pieces were imbedded in paraffine and serial sections made through the entire cord and medulla oblongata. Parts of all convolutions of the cerebrum and the subcortical ganglia have been examined.

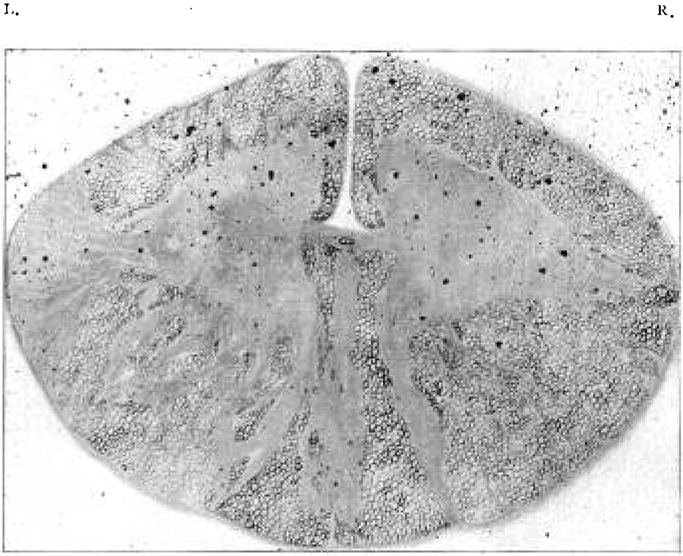

FIG. I. Cross-section through the second cervical segment.

For practical reasons I will describe the microscopical condition of the spinal cord from above downward, beginning just below the cervico-medullary junction.

Second cervical segment. The left anterior horn shows a marked shrinkage, the lateral diameter being half that of the right horn. There are no ganglionic cells in the left horn at all, except a very few highly atrophied cells at the region of Stilling's nucleus. On the right side the different groups of ganglionic cells are all present, but also in a condition of atrophy. They are all considerably shrunken and in a chromophilic condition. The pericellular spaces are enlarged. There is a proliferation of blood-vessels in the gray substance, and the perivascular spaces are considerably enlarged. The walls of the blood-vessels, however, are of their normal thickness and show no changes whatsoever.

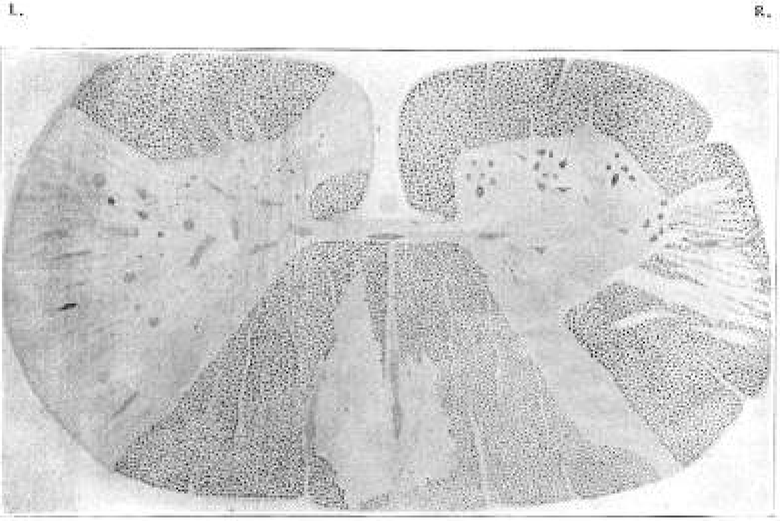

FIG. II. Cross-section through the sixth cervical segment.

The white substance is normal with the exception of the columns of Goll, where there is an irregular field of proliferating connective tissue extending from the center between the gray matter and the pia towards the periphery.

Third and fourth cervical segments.—There is a proliferation of connective tissue starting from the left anterior horn, extending irregularly into the white substance. Otherwise the conditions are the same as in the second cervical segment.

Fifth cervical segment.—The left anterior horn is still considerably smaller than the right one. The gray substance is found to be very vascular, and there are a great many granular cells scattered about, while the left horn has only very few highly atrophied cells belonging to the medial group. The cells of the right horn show only slight changes. They are of about normal size, but are mostly homogeneous. Nissl's granules have evidently disappeared. The axis-cylinder is broken off in some cells, and in other instances they appear to be swollen. The proliferation of connective tissue starting from the left horn into the white substance is more marked than in the previous segment. The area of connective tissue in the columns of Goll is also the same as before, and contains many blood-vessels.

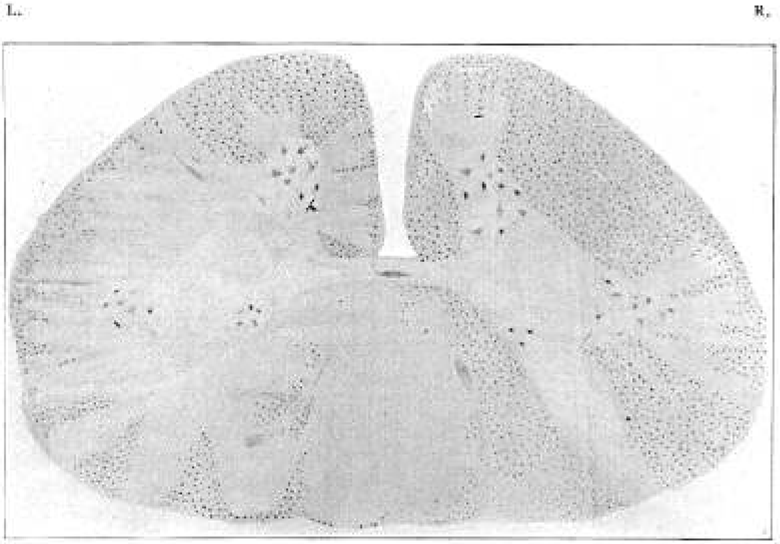

FIG. III. Cross-section through the first dorsal segment.

Sixth cervical segment.—The right horn contains the different groups of ganglionic cells, but they are nearly all atrophied. A good many cells contain a considerable amount of pigment. The proliferation of connective tissue from the left horn has spread fan-like through the entire white substance to the periphery, destroying the white substance altogether. A smaller fan-like proliferation spreads from the internal anterior corner of the left horn through the anterior columns of the white matter. On the right side there is a smaller proliferation of connective tissue starting from the right anterior horn, so that both horns give the appearance of a lateral enlargement. The blood-vessels appear very numerous in both the gray and white substance. They are full of blood, and the perivascular spaces are considerably enlarged. The posterior columns show a large area of connective tissue covering nearly the entire dorsal half of the columns of Goll. Here we also have the same proliferation of blood-vessels, around which there are many granular cells.

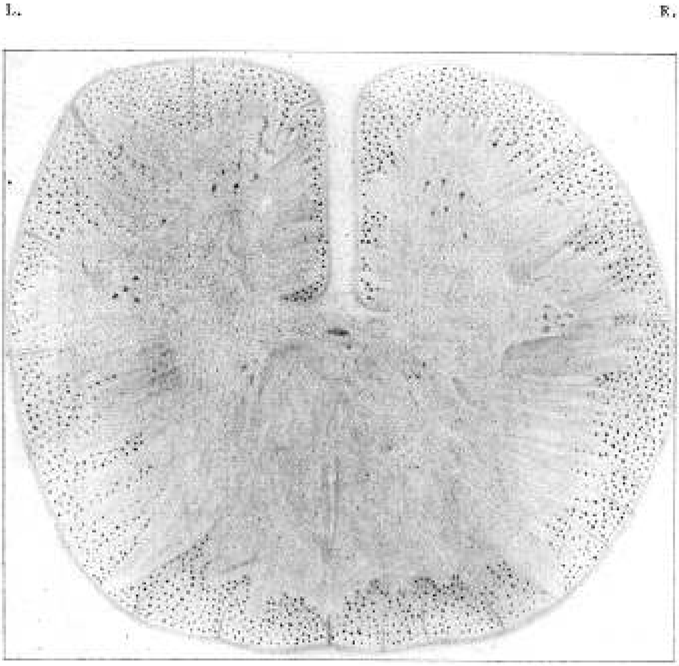

Fig. IV. Cross-section through the seventh dorsal segment.

Seventh cervical segment.—There are very few ganglionic cells in the left horn, and those in the right horn are all atrophic. The fan-like proliferation of connective tissue from both horns has become larger. Of the posterior columns, those of Goll are entirely converted into connective tissue, while those of Burdach look more normal, otherwise the same as before. There are no normal cells in either horn, except a few in the lateral group of the right side amidst the proliferating connective tissue. On both sides there is a proliferation of connective tissue from the anterior internal corner of the anterior horn through the white substance to the periphery, otherwise the same as before.

Eighth cervical segment.—On the right side all the different groups of ganglionic cells are present, and they are perfectly normal; only a few show a slight atrophy. On the left side there are more atrophic cells than on the right, but still there are some normal cells here, too, and the lateral group especially seems to be normal. We find here some normal cells within the fan-like expansion of connective tissue. The atrophic cells all contain a considerable amount of granular pigment. The blood-vessels are enormously enlarged and increased in number. But nowhere are there any changes in their walls.

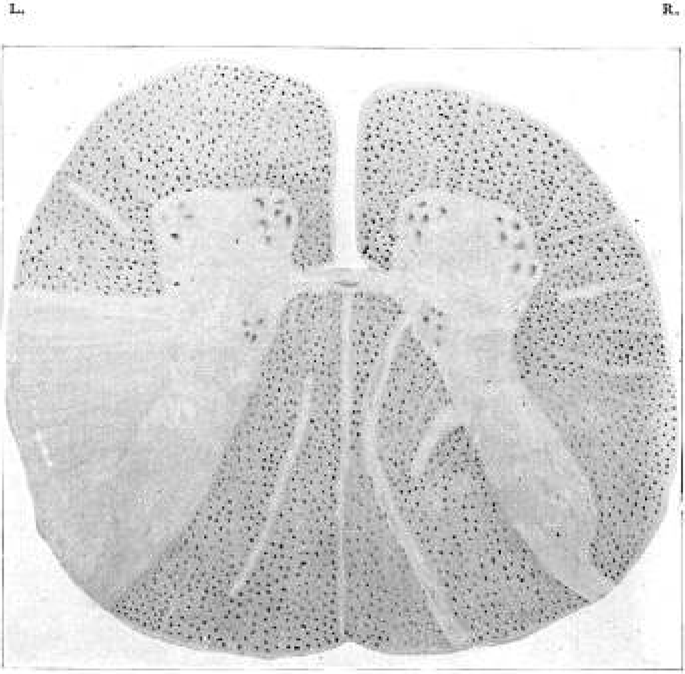

FIG. V. Cross-section through the second lumbar segment.

First dorsal segment.—The ganglionic cells on both sides in the different groups in the anterior horns and in the columns of Clarke, are mostly atrophic; only here and there we find some normal cells. The fan-like proliferation of connective tissue on the left side embraces the entire half of the cord, with the exception of a small area of the anterior white column. On the right side, this proliferation has become less, so that we have considerably more normal white fibers. The posterior columns are converted into connective tissue, with the exception of the larger part of the right column of Burdach.

From the second to the sixth dorsal segment we have about the same condition as in the first dorsal segment, the normal ganglionic cells alternating with highly atrophic ones, and the expansion of the areas of the proliferating connective tissue in the white substance varying on the different levels. The proliferation of blood-vessels remains very considerable, and also the enlargement of the perivascular spaces. At some parts we find a considerable accumulation of granular cells.

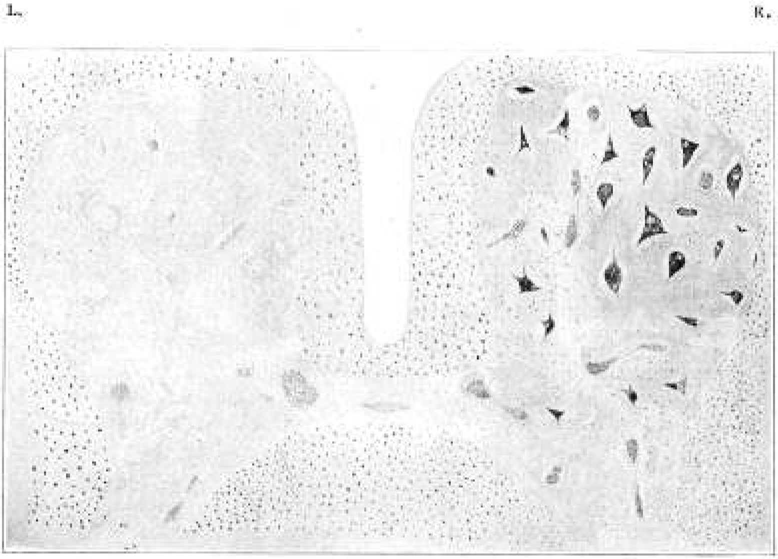

FIG. VI. Cross-section through the fourth lumbar segment.

Seventh dorsal segment.—We find nearly the entire cord converted into connective tissue, so that it has an aspect of a transverse myelitis, only at the periphery all around the section there are normal nerve fibers; the outlines of the gray substance being difficult to recognize. The degree of atrophy of the nerve cells seems not to be in proportion to the pathological process of the surrounding tissues. We find sometimes perfectly normal cells surrounded by connective tissue.

The lower we go down in the cord the larger grows the normal space at the periphery.

Ninth dorsal segment.—The white substance has again become normal with the exception of a small portion at the left side, where a fan-like proliferation of connective tissue extends again from the anterior horn to the periphery. The columns of Goll also are converted into connective tissue. The gray substance is destroyed nearly altogether, and only a very few cells are present.

Tenth dorsal segment.—Again the entire cord is converted into connective tissue, with the exception of a small part of the periphery.

Eleventh dorsal segment.—There are very few cells in both anterior horns, while the lateral horns and the columns of Clarke are nearly normal. The white substance has become more normal again, especially on the right side, while the left side is, however, destroyed, and the posterior columns show nearly normal fibers at the lateral margins.

Twelfth dorsal segment.—The proliferation from both horns is increased again, so that on both sides the white columns appear to be destroyed. There are only normal white fibers in the anterior columns The cells in the anterior horns and columns of Clarke are present, although highly atrophic.

First lumbar segment.—First lumbar segment shows the same conditions as the twelfth dorsal.

Second lumbar segment.—The white substance on both sides is again more normal, with the exception of a large fan-like proliferation of connective tissue on the left side.

Third lumbar segment.—On the right side the white substance seems to be entirely normal, while on the left side there is still a considerable area of connective tissue. The posterior columns have become normal. The cells are all atrophic. In the left horn the cells seem to have disappeared altogether.

Fourth lumbar segment.—The white substance has become perfectly normal, with the exception of a very small area on the left side. The ganglionic cells on the right side are large in size, but contain an unusual amount of pigment. A good many cells are chromophilic, and sometimes the axis-cylinder seems to be broken off. On the left side there are no cells at all, with the exception of a very few highly atrophied remnants. There are a good many empty spaces, indicating the disappearance of the cell. We find the same condition in the fifth lumbar segment.

Towards the lower part cells appear again on the left side. The white substance has remained normal. The sacral columns appear normal, with the exception of some atrophic cells.

In the first sections of the medulla oblongata, we find the same proliferation of connective tissue in the ventral part, affecting mostly the pyramidal tracts, sometimes extending dorsad into the lemniscus and the substantia reticularis alba. The nucleus of the hypoglossus contains many atrophic cells on both sides. On the left side the proliferation of connective tissue occupies a field which is limited dorsad by the substantia gelatinosa and the nucleus of the hypoglossus; laterally by the nucleus lateralis. Besides the nucleus of the hypoglossus, those of the glossopharyngeus and vagus and the nucleus ambiguus contain many atrophic cells, while these nuclei on the right side are normal. At the level where the fibers of the glossopharyngeus begin to leave the medulla, everything has become normal, and remained so up to the level of the nucleus of the oculomotorius. The only changes are enlargement of the perivascular spaces, not only in the medulla oblongata, but also in all parts of the brain. The cell layers of the cortex, over all parts of the brain have been carefully examined and have been found perfectly normal.

A number of interesting questions arise from these anatomical findings. The most important, of course, is the diagnosis. When I presented the patient to the Neurological Society three years ago, I had made the diagnosis amyotrophic lateral sclerosis, maintaining that the pathological process had started from the left anterior horn of the cervical part of the cord and proceeded from there, first to the left pyramidal tract, then to the right horn and right pyramidal tract. The microscopical examination shows this assumption to be true. A glance at the cervical part of the cord will show an anatomical condition of this kind. Still, are we justified in calling this a case of amyotrophic lateral sclerosis? The clinical course of the disease would surely be in favor of it. We had the typical three stages which Charcot described: the atrophy, the rigidity with the muscular contractures and the bulbar symptoms. The rapid course of the disease, which lasted only a few years, would also correspond to Charcot's description. The pain which the patient experienced towards the end of his life does not, of course, belong to the disease, but could be explained somehow or other by the muscular contractures or some complication. From the anatomical findings, however, the diagnosis of amyotrophic lateral sclerosis cannot be sustained. Whether amyotrophic lateral sclerosis is a *morbus sui generis*, which is to be distinguished from progressive muscular atrophy and bulbar paralysis, or whether, as many authors maintain, these diseases form only different stages or degrees of one and the same disease, is a question which cannot be discussed here. Whatever view, however, we might hold regarding this question, one must regard amyotrophic lateral sclerosis, whether it forms a separate disease or not, as a systemic disease; like progressive muscular atrophy, a primary degeneration of the motor neurons.

To avoid misunderstanding, it would be still better to adopt Dana's suggestion and change the name systemic disease into neuron disease. The important question then in this special case would be: have we to deal with a neuron disease, that is, a primary degeneration of a certain group or groups of neurons belonging to one system, or with some other pathological process through which the neurons have become affected secondarily? There can be no doubt that we have to deal here with the latter condition. In favor of this view are:

First, the anatomical process itself. There is at several levels of the cord, but especially at the lower cervical and upper dorsal region, a fan-like proliferation of connective tissue from the gray substance, which we do not see in primary degeneration of the white columns. It seems evident that this proliferaion of connective tissue has destroyed the white substance, but has not been produced through the degeneration of the latter, as is the case in primary neuron diseases. The enormous proliferation of blood-vessels, as well as the considerable enlargement of the perivascular spaces, do not indicate a primary degeneration of the neurons.

Secondly, the topography of the pathological process. The inflammatory process which apparently originated in the gray substance, spread in all directions and involved all parts of the white substance without any regard to systems. We find the columns of Burdach and Goll and the anterior columns affected, as well as the pyramidal tracts; that the inflammation did not follow any systemic paths, but spread from a central focus in all directions, is especially evident in the dorsal region, where the outlines of the gray substance have become very indistinct and the inflammatory field occupies nearly the whole cord, leaving some normal fibers only around the periphery, regardless of the system to which they belong.

Thirdly, The atrophy of the ganglionic cells is not in proportion to the changes of the surrounding tissue. We find sometimes normal or only slightly atrophied cells amid the most remarkable changes, so that the affection of the nerve cells must be regarded as a secondary matter.

Therefore, we have to deal here with a case of interstitial myelitis, probably of vascular origin, in which the nerve cells have become atrophied secondarily.

As to the nature of systemic or neuron diseases and their relation to other inflammatory processes of the central nervous system, there seem to be some discrepancies among different authors. There is on the one hand a tendency to deny the existence of systemic diseases altogether. The supporters of this view maintain that all systemic diseases, including tabes and progressive muscular atrophy, consist of myelitic processes by which the neurons are affected secondarily. On the other hand, there are some followers of the theory of systemic diseases who draw very narrow limits to the interstitial myelitic processes, considering, for instance, poliomyelitis anterior a systemic disease, as was first suggested by Charcot.

Recent anatomical investigations of fresh cases of infantile spinal paralysis, as well as the generally accepted theory of its infectious nature, have put it, I think, beyond doubt that we have to deal here, not with a systemic disease, but an interstitial inflammatory process. The fact that the anterior horns are always the main seat of the affection is due to the more abundant blood supply of these parts. The statement of some authors that certain groups of ganglionic cells are generally more affected by the disease than others has not been confirmed by closer investigation. On serial section, some groups appear to be affected at some level and intact at another. The atrophy of the cells is dependent upon the distribution of the smaller branches of the central vessels, and the fact that in one section some cells are atrophic, while others are intact, is due to the difference in the blood supply, the different groups of cells belonging to different areas of blood-vessels.

As far as spinal diseases of later life are concerned, we must admit that in many cases it is not only extremely difficult, but sometimes quite impossible, to distinguish between a systemic or neuron disease and a myelitis merely from the clinical symptoms. My case is a good illustration of this fact. Every-body who saw the case agreed that it was a systemic disease; that is, either progressive muscular atrophy or amyotrophic lateral sclerosis, while the microscopical examination revealed quite different conditions. A myelitis might possibly simulate any neuron disease.

I am inclined to believe that quite a number of the cases which have been reported as having developed from an infantile spinal paralysis to a progressive muscular atrophy, after a microscopical examination, would prove to be, like my case, instances of myelitis, instead of primary neuron disease.

The next question would be whether this man's infantile spinal paralysis had any relation to his later disease, and if so, of what nature was this relation? I have mentioned the different theories which have been advanced on this subject. Most of them are based on the theory that poliomyelitis anterior is a systemic disease and could not therefore be applied to this case.

The third theory, however, would correspond to the clinical and anatomical condition of this case; that is, that the old scar which was produced by the inflammatory process in the gray matter of the anterior horns, formed a latent but permanent inflammatory focus, which at any time might light up afresh and cause a new set of symptoms. That in my case the old scar in the left anterior horn of the cervical part of the cord formed the starting point for the disease was diagnosed during life, and has been confirmed by the autopsy. Whether the old scar contained the poison to set up the new disease, or whether we have to deal with a new infection which attacked the old scar first, as the *locus minoris resistentiæ*, is, of course, a question which cannot be decided with absolute certainty. I think, however, that there is no necessity of assuming a new infection. It is perfectly possible that, as Strümpell says, the virus in the old scar behaves like an encapsuled tubercular focus, remaining dormant for a long time, and then setting up a new inflammation.

Another point of interest is whether this case could contribute to our knowledge regarding the functions of the cells of the spinal cord. Cases of infantile spinal paralysis, which have left behind atrophies of isolated muscles, and which come to autopsy in later life, are especially appropriate for this study; in fact, they equal in many respects an experiment. Naturally, such cases are very rare. Charcot reports that he saw the scar of infantile spinal paralysis in the anterior horns as long as sixty years after the disease, but a special study of the cells as to their function was not made by him. A very valuable contribution to this part of science has been furnished by Collins, who had the chance of examining a spinal cord of a man who died from a nephritis, and who had an attack of infantile spinal paralysis twenty-three years previously. Collins was able to corroborate by his case the views regarding the nucleus of the upper extremity. This nucleus is composed of three groups of cells located in the lateral part of the anterior horns, and extending from the lower part of the fourth cervical into the first dorsal segment. Collins was furthermore enabled to show by his case that a group of cells belonging to this upper-extremity nucleus, being located at the seventh cervical segment and being arranged internally, governed the function of extending the fingers; while another group of cells between the last cervical and first dorsal segments, being arranged laterally, is concerned in supplying the muscles that cause flexion of the fingers.

My case is not quite as appropriate for this study, because the man developed another spinal disease, which made it rather difficult to distinguish between the old and recent affection, while Collins' patient exhibited the spinal cord of the old infantile paralysis without any further changes. There are, however, some valuable facts which can be utilized in spite of this. In order to compare the anatomical findings with the clinical symptoms, I may be allowed to recall with a few words the progress of muscular disease in the patient's upper extremities.

His left arm remained in a highly atrophied condition after the infantile disease. The muscles of the shoulder and upper arm were involved most. The muscles of the hand had remained perfectly normal. During the course of the later disease the whole left extremity became gradually paralyzed, the only movement which the man could perform being a slight flexion of the fingers. The right upper extremity, which had not been affected at all by the infantile paralysis, also became paralyzed during the later disease, but here only the movement of the arm had been lost; the hand could be moved perfectly well in spite of a considerable atrophy of the interossei. The microscopical examination revealed an entire destruction of the upper-extremity nucleus on the left side, with the exception of a few cells at the outer margin of the left anterior horn in the eighth cervical segment. If we hold these cells responsible for the limited movement of flexion which the man could perform, we can corroborate Collins' statement as to his outer group in this respect.

On the right side the upper-extremity nucleus was destroyed down to the middle part of the seventh cervical segment. At this level the cells of this nucleus appeared normal, with the exception of the anterior group, which was missing. With the beginning of the eighth segment, however, this group also became normal, so that the entire nucleus appeared in good condition down to the beginning of the first dorsal segment. This is in perfect harmony with the unimpaired movements of the man's right hand, the atrophy of the interossei being due to the atrophy of the cells in the first dorsal segment.

The atrophy of the cells which innervate the muscles of the back, *i. e.*, the group of cells which we located internally in the anterior horns, accounts for the man's inability to sit up or hold up his head.

There seems to be a discrepancy in the findings of the cortical cells between this case and the one of Collins. While in my case I could not detect any abnormal conditions in the cortical areas corresponding to the part which was affected by the infantile paralysis, Collins reported an atrophy both in size and constituents of this area. This, however, is due to the fact that my patient, in spite of the atrophy, could move and use his extremity, while in Collins' case the affected extremity was perfectly motionless. This difference between the two cases would, therefore, corroborate Collins' theory, that not the atrophy but the inactivity produces the affection of the other group of neurons. The fact that in my case the cortical cells of the cortico-spinal neurons were perfectly normal, in spite of the severe affection of the pyramidal tracts, is another support of the statement that we have to deal in this case, not with a systemic or neuron disease, but with a local inflammatory process, *i. e.*, a myelitis.

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84. EINE NEUE FÄRBUNG FÜR DAS NERVENSYSTEM (A New Stain for Nervous Tissue). P. Kronthal (Neurologisches Centralblatt, 18, 18 9, p. 196).

Kronthal has described a new method of treating nervous tissue which permits of the study of the nerve cells as well as the fibers. It combines the method of Golgi and that of Weigert in specimens which are successfully treated. He first hardens the nervous tissue, perfectly fresh, in a mixture consisting of equal parts of a saturated aqueous solution of the formate of lead and 10 per cent formalin. The pieces remain in this fluid five days at the ordinary temperature. They are then transferred, without washing, into a mixture of equal parts of 10 per cent. formalin and hydrogen sulphide solution (a saturated solution of sulphureted hydrogen in water), in which mixture they remain five days. They are then washed, dehydrated in alcohol, imbedded in celloidin, and the sections cleared in xylol-carbolic solution and mounted in balsam with cover. According to the author's experience, they suffer no change after at least ten months.

For the conservation and staining of an entire brain, the author has tried the following procedure: The brain was placed for 14 days in 2½ liters of 1 per cent. formalin and saturated solution of formate of lead equal parts of each; and then in 2½ liters of 10 per cent. formalin and 2½ liters of hydrogen sulphide solution, and allowed to remain in this for 14 days. It was then kept in a solution of equal parts of glycerine and 90 per cent. alcohol until it was ready for sectioning. The glycerine, while making the brain tissue a little softer, makes it more elastic. Brains kept in alcohol alone are too brittle for large sections.

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